The rate of facultative sex governs the number of expected mating types in isogamous species

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It is unclear why sexually reproducing isogamous species frequently contain just two self-incompatible mating types. Deterministic theory suggests that since rare novel mating types experience a selective advantage (by virtue of their many potential partners) the number of mating types should consistently grow. However, in nature, species with thousands of mating types are exceedingly rare. Several competing theories for the predominance of species with two mating types exist, yet lack an explanation for how many are possible and in which species to expect high numbers. Here, we present a theoretical null model that explains the distribution of mating type number with just three biological parameters; mutation rate, population size and the rate of sex. If the number of mating types results from a mutation-extinction balance, then the rate of sexual reproduction plays a crucial role. If sex is facultative and rare (a very common combination in isogamous species), mating type diversity will remain low. In this rare sex regime, small fitness differences between the mating types lead to more frequent extinctions, further lowering mating type diversity. We also show that the empirical literature supports the role of drift and facultativeness of sex as a determinant of mating type dynamics.

In most sexually reproducing species, gametes do not fuse indiscriminately: syngamy only occurs between gametes of complementary mating types. The evolution of gamete self-incompatibility (SI) is puzzling, as self-discriminatory mutants limit their reproductive opportunities. A number of theories for SI have been proposed (review: [7]), including modifying the costs of sex by promoting out-crossing, maximizing the rate of attraction between gametes, and managing conflict between cytoplasmic organelles.

Suppose that SI evolves by one of the selective forces above. The next question that arises is How many mating types might we expect to see? A simple answer is very many: any novel type that can fuse with the entire resident population is favoured when rare (negative frequency-dependence) and thus will always invade [35, 48]. Extrapolating this to the extreme predicts a population with as many mating types as individuals. However, such abundances are not observed in the natural world. While examples of species with numerous mating types exist (the fungus *Schizophyllum commune* has over $23,000$ [38]), the vast majority of species feature only two [7], contradicting our naïve extrapolation above.

A more thorough treatment requires considering gamete morphology. The morphological similarity between gametes in isogamous species [42] contrasts with the clear dimorphism (sperm, eggs) in anisogamous and oogamous species. Explanations for the transition from isogamy (considered the ancestral state [6, 16, 26, 65]) to two sexes (anisogamy) usually consider competition for fertilization driving disruptive selection ([41, 58, 64]). If within-sex mating (egg-egg or sperm-sperm syngamy [31]) is detrimental, selection favours linkage between mating types and sexes [55], and it becomes difficult to envisage how a novel mating type could benefit by mating with more than half the population. Conversely, isogamous species should not *a priori* feature this fundamental restriction; indeed *S. commune* is isogamous. Yet, the majority of isogamous species have just two mating types [22].

Our aim is to explain the preponderance of species with very few mating types, as well as the existence of species with many more, in isogamous populations. Currently, numerous competing theories exist (reviews: [7, 36]), often invoking the same hypothetical mechanisms that generate SI. One theory posits that two types maximize the efficiency of pheromone signalling while preventing counterproductive attraction to own pheromones [22, 30, 31]. While plausible [24], this hypothesis does not account for exceptions [59] and does not predict precisely when more than two mating types might be expected. Furthermore, the predominance of two types remains somewhat unaccounted for under these models, as the conditions that prevent the emergence of three types appear stringent [24]. Alternatively, uniparental inheritance (UPI) of organelles has been suggested to drive the evolution of mating types [32–34], with the directionality of inheritance (donor and receiver) limiting the number of mating types. However recent theoretical work accounting for frequency-dependent fitness in UPI has demonstrated that selection for UPI does not obviously lead to the evolution of mating type number [23]. A further issue is that the hypothesis does not address the mating type num-

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bers in species without UPI. A similar lack of predictive power applies to the idea that evolutionary constraints on genome architecture might make it difficult for a new type to arise that can mate with others [52, 69].

Verbally, it has been argued that genetic drift may limit the number of mating types [35]. Here we model the relevant population genetics. Crucially, we insert an overlooked biological component to previous verbal ideas [7, 35]: we take explicitly into account that the majority of isogamous sexual organisms engage in facultative sex. We show that the consequent alternation of asexual and (rare) sexual cycles has a major effect on mating type dynamics. We derive the expected number of mating types when genetic drift, causing extinction of types, is balanced by mutations, which introduce new mating types to the population.

I. RESULTS

A. Model

Our model aims to be general by being simple. However, we contextualise it with model isogamous organisms, such as the single-celled green alga *Chlamydomonas reinhardtii* and *Saccharomyces* yeasts. Consider *Chlamydomonas*. In the wild, *C. reinhardtii* mostly exists in a haploid state, replicating asexually through mitosis. Falling nutrition levels instigate the sexual phase of its life cycle [67]; facultative sex under stress is common across lower eukaryotes [50]. The haploid cell mitotically divides into four gametes [27, 68]. One of two alleles at a single locus determine the mating type [19]. Opposite mating types engage in syngamy. Following meiosis the cells divide, with half inheriting the mating type of each parent.

We take a population genetics approach and use a Moran-type model with a constant population size N (birth-death events are coupled) and overlapping generations [29]. SI mating types are determined by an allele at a single locus. In order to explore the dynamics of mating type number, we allow for an infinite number of mating type alleles at this locus. For the full approach see Methods; here we review the salient points.

We denote by α_i mating type i, with number of individuals n_i and frequency $x_i = n_i/N$. The number of mating types present in the population is denoted M. Individuals can experience three classes of event: asexual reproduction, sexual reproduction and mutation. Both types of reproductive events produce a single progeny; for sexual reproduction, the progeny inherits either parents genotype with probability 1/2 (see Fig. 1). The parameter c controls the rate of sex from $c = 1$, entirely asexual reproduction, to $c = 0$, obligate sex. Our assumption of a constant propensity for sexual reproduction $(1 - c)$ means we consider the many mechanisms for the evolution of recombination rates [28] beyond the scope of our model. For instance, in the *Chlamydomonas* example, c can be interpreted as the probability that an individual is in a non-stressed state and reproduces asexually.

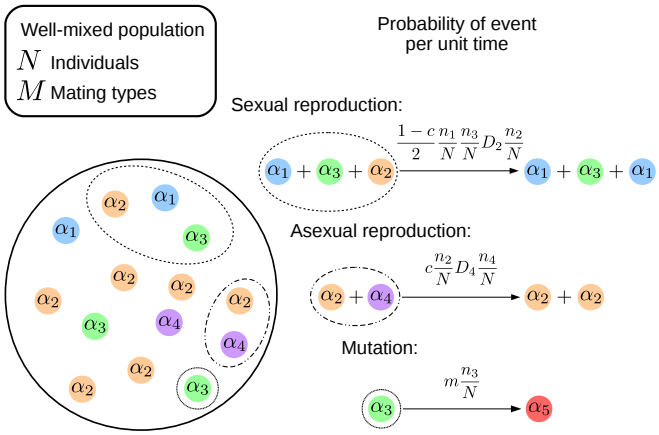

FIG. 1. Visualization of the model illustrating three types of potential event; sexual reproduction, asexual reproduction, and mutation. Each occurs with a probability rate proportional to the frequency of each type involved in the event. In this example, type α_2 is less likely to reproduce sexually than the other types due to its high frequency. However, as it can engage in asexual reproduction, its frequency can still potentially increase due to drift. At a given time there are M mating types present in the population. All events (a total of $M^2(M-1)$ for sexual reproduction, M^2 for asexual reproduction and M for mutation) lead to one of M^2 different transitions in which one mating type increases and another decreases by one. Summing over all events that lead to each transition yields Eqs. (1-2).

Mutants arrive independently at a rate m, are novel to the population, SI, and mate with resident types at the same rate as resident SI types mate with each other. This liberal assumption does not account for maladaption in signaling or syngamy of the mutant with its SI ancestor. However, since identical fitness across all mating types is an unlikely scenario [60] (see also [57] in the context of sex-ratio evolution), we consider an extension of our main model, in which each type has its own mortality rate, D_i, drawn chosen from a normal distribution with mean one and variance σ.

Since each event involves replacing one individual with another, we combine multiple events into a single term, $\mathcal{T}_{ij}$, the probability per unit time of n_i increasing by one and n_j decreasing by one;

$$\mathcal{T}_{ij} = \left[\overbrace{c\frac{n_i}{N}}^{\text{Asexual reproduction}} + \overbrace{\left(\frac{1-c}{2}\right)\frac{n_i}{N}\left(\frac{\sum_{k\neq i} n_k}{N}\right)}^{\text{Sexual reproduction}} \right] \left[\overbrace{D_j \frac{n_j}{N}}^{\text{Death}} \right] \quad \text{if} \quad n_i > 0\,, \tag{1}$$

$$\mathcal{T}_{ij} \propto \left[\underbrace{m\frac{n_j}{N}}_{\text{Mutation}} \right] \quad \text{if} \quad n_i = 0\,. \tag{2}$$

Our model includes ideas from Fisherian sex ratio theory (common types have lowered mating success) while re-

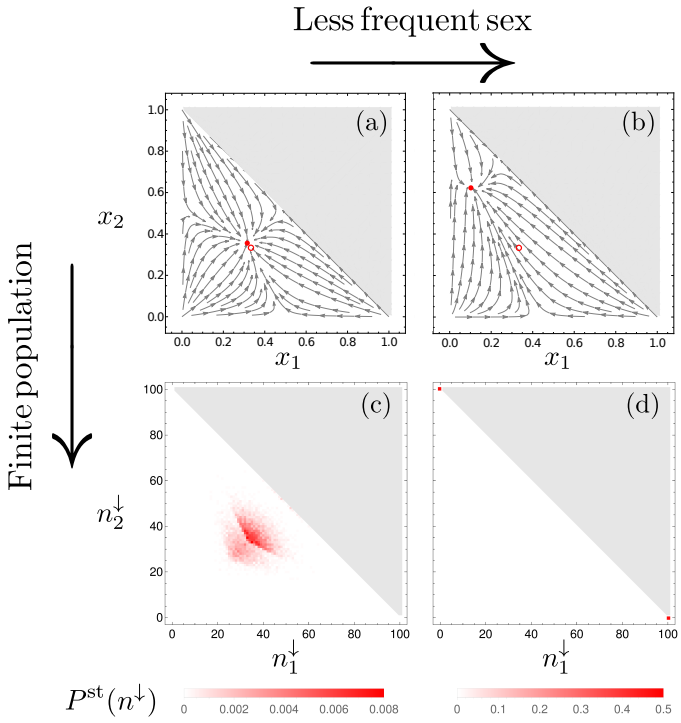

FIG. 2. Dynamics of mating type frequency with initially three mating types ($M_0 = 3$). Plots (a) and (b) are phase plots of the dynamics in the deterministic limit ($N = \infty$, see Eq. (8)) while (c) and (d) show the results of stochastic Gillespie simulation of Eq. (2) ($N = 10^3$), with the stationary distribution $P^{\rm st}(\boldsymbol{n})$ projected into the 2D plane. In the presence of weak selection ($\sigma = 0.04$, plots (a-d)), the fixed point of the deterministic dynamics [(a) and (b) red disk, see Eq. (11)] is shifted from the neutral prediction $x_i = 1/M_0$ (red open circles). This shift is greater when sex is less frequent [$c = 0$ in plots (a,c), $c = 0.8$ in plots (b,d)]. In (c,d), drift allows the system to move away from the deterministic fixed point. When sex is rare, both drift and weak selection increase the extinction rate (see plot (d), where only a single mating type allele is present at long times). In plots (c) and (d), $m_g = 5 \times 10^{-3}$.

laxing the assumption, inherent in classic Fisherian theory, that failure to engage in sex implies complete reproductive failure (here asexuality is still an option). The lowered mating success of common types is taken into account by the term $\sum_{k\neq i} n_k$; sex can only occur between different types. Rarer mating types will have an increased per-capita probability of participating in sexual events compared to more common types, while there is no difference in the context of asex. The strength of selection against common types (negative frequency dependence) therefore depends on the frequency of sex.

B. Model behaviour

We first consider an infinite population with rare mutations ($m \ll 1$) and no differential mortality ($\sigma = 0$). Denoting by M_0 the initial number of mating types present, the population approaches a state where all types are equally represented ($x_i = 1/M_0$, see Fig. 2a). It resides here until mutation introduces a novel mating type; a new stable state then emerges at $x_i = 1/(M_0 + 1)$. This pattern leads to linear growth in the number of mating types (see Eq. (10)) and predicts, at very long times, infinitely many mating types each at infinitely low frequency (see Fig. 3a), in agreement with earlier simpler models [35]. We next allow differential mating type mortality ($\sigma > 0$). Types are no longer equally represented (see Eq. (11) and Fig. 2a-2b). The departure from even type frequencies increases with M (the number of mating types), c (the rate of asexual reproduction) and σ (mortality variance). The probability that this polymorphic equilibrium is stable also decreases with these parameters (see Fig. 3b), limiting the number of mating types to a large but finite value.

Turning to finite populations, an infinite number of mating types becomes obviously impossible, even in the absence of mortality differences between the mating types. The number is instead determined by a balance between mutation and extinction. Low mutation rates (that limit the supply of new types) and population sizes (that increase drift) reduce M. These relatively obvious effects co-occur with the more interesting effect of a nonzero propensity for asexual reproduction, c. High c can greatly amplify the effect of drift which, by speeding up the extinction rate, leads to fewer mating types (see Fig. 3c). Differential mortality rates exacerbate this process by shifting the equilibrium deterministic fixed point closer to the extinction boundaries (see Fig. 3d).

The above evokes very long term arguments, and we seek an analytic characterization of the populations behaviour once any transient dynamics have died away. The mathematical analysis is conducted under the assumption that there are no fitnesses differences between mating types ($\sigma = 0$); the dynamics outside this regime are explored via simulation.

Denote by $\mathcal{P}^{\rm st}(M)$ the probability of observing M mating types in the population at infinite time and assume no differential mortality. Employing approximations that rely on biologically reasonable assumptions of large N and small $m_g = mN$ (novel mating types arise far less than once per generation), we can obtain bounds on the mode of $\mathcal{P}^{\rm st}(M)$ under three different scenarios in which all mating types have the same mortality; for obligately sexual ($c = 0$), obligately asexual ($c = 1$) and facultatively sexual ($1 > c > 0$) organisms. For obligate sex, we find

$$\sqrt{\frac{N}{\mathrm{W}\left[\frac{1}{4\mathrm{e}^2\mathrm{m}^2\mathrm{N}}\right]}} > \mathrm{Mode}\left[\mathcal{P}^{\rm st}(\mathrm{M})\right] > \sqrt{\frac{-\mathrm{N}}{2\left(1+\log\left[2\mathrm{m}\right]\right)}}, \tag{3}$$

where $W[z]$ is the Lambert W function [1]. Thus, for obligately sexual isogamous organisms, we predict many mating types, of the order hundreds (see Fig. 4).

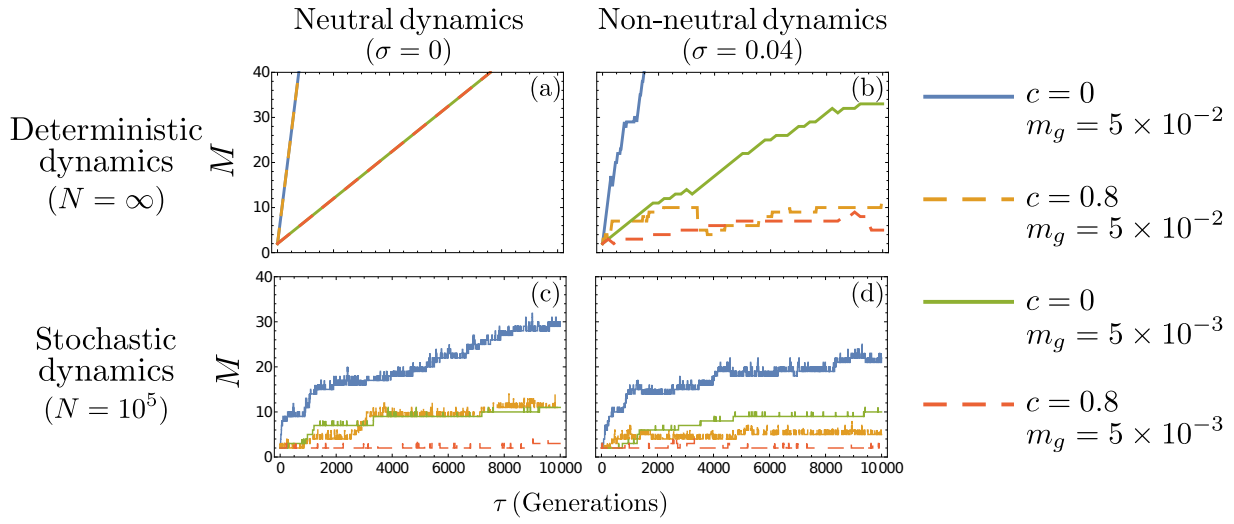

FIG. 3. Dynamics of the mating type number, M, with $M_0 = 2$ for various parameters (coloured and dashed lines) under different modelling assumptions [plots (a-d)]. In the infinite population size limit when all mating types are equally fit [plot (a)] the rate of asexual to sexual reproduction, c, does not effect the dynamics of M (see Eq. (10)). However, when weak fitness differences between the alleles are present [plot (b)], rare sex (large c) decreases the strength of selection for even mating type number (see Fig. 2), leading to lower values of M (dashed lines). Finite population sizes [plots (c) and (d)] further lower mating type numbers through drift-induced extinctions. In this context, lower mutation rates also limit the observed number of mating types.

For obligately asexual organisms, we find

$$\text{Mode}\left[\mathcal{P}^{\text{st}}(\text{M})\right] \approx 1\,; \tag{4}$$

If sex is facultative (the most important category for isogamous organisms), the expected number of mating types in the population lies between the upper bound in Eq. (3) and the lower bound in Eq. (4). A precise solution can be obtained numerically (see Eq. (17) and Fig. S8), leading to our most important finding (Fig. 4): at low but non-zero rates of sexual reproduction $(1 - c)$, large populations can maintain distinct mating types (unlike the asexual system) but the number of mating types can be very low (unlike the sexual system).

Mortality differences ($\sigma > 0$) prevent us from obtaining analytical results of the type described above. Simulating the model, we find that $\sigma > 0$ decreases the expected number of mating types for all c (see Supplementary Information). This decrease is larger when c is large (sex is rare) as this leads to more distorted mating type frequencies (see Eq. (11)) and consequently more frequent extinctions.

C. Empirical support for model

We predict low numbers of mating types to associate with small effective population size (N_e), low mutation rates and rare sex. Before evaluating the relevant evidence, we first list why estimating these parameters is challenging.

that is, we expect only a single, non-expressed (in the absence of sex), mating type. If mutants arise much less than once per generation (our assumption), genetic drift purges these neutral variants from the population faster than they are produced.

Estimating mutation rates producing new mating types is particularly difficult, as we are unaware of a single documented case of a *de novo* mating type arising. Thus, like Wright [72] we simply discuss what is reasonable.

The second parameter, N_e, typically falls below the census population size as various processes (local population structure [56], bottlenecks, and sexes and recombination themselves [10]) can accentuate genetic drift. Though species-specific estimates of N_e vary, we present results based on N_e ranging between 10^6 and 10^7 a compromise that is both evidence-based [2, 20] and avoids interpreting model performance too optimistically (e.g. stress induced sex coinciding with population bottlenecks could lower N_e, yielding a better match between model predictions and observations).

Finally, many otherwise well studied species are data deficient for their rate of sex. Estimates based on molecular methods [66] are disputed [53]; data on wild populations [12, 43] are rare. Sex occurs in many organisms previously thought to be asexual [40], and cryptic sex is common [15]. We focus on well studied species out of necessity, noting this may overestimate the frequency of sex: organisms may become model species precisely because sex is straightforward to induce in laboratory settings. When evaluating the propensity for sex in our model $(1 - c)$ empirically, a difficulty is that we predict

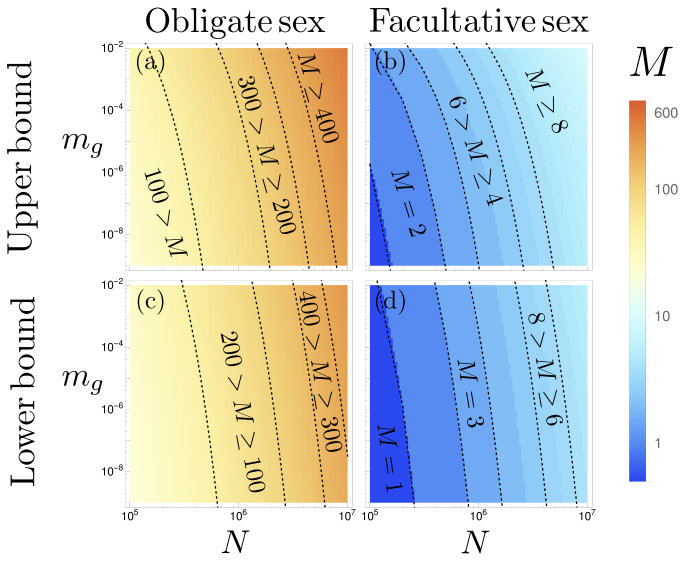

FIG. 4. Bounds on the expected number of mating types predicted by the model as a function of the population size, N, and the per-generation mutation rate, m_g when there are no selective differences between the mating types ($\sigma = 0$). Results shown for an obligately sexual population ($c = 0$) and a facultatively sexual population in which sex is rare ($c = 0.999$). Bounds are obtained by evaluating Eq. (3) in the obligately sexual case (plots (a) and (c)) and numerically solving Eq. (17) in the facultatively sexual case (plots (b) and (d)).

lower c to yield more mating types (higher M); simultaneously, higher M can lead to more mating opportunities and thus amplified signatures of sex. However, disentangling these features is possible: increasing the number of mating types should lead to modest changes in the amount of sex (matings are possible between $50 - 100\%$ of the population); larger differences reflect actual differences in propensity for sex, $(1 - c)$. We proceed with the above caveats in mind.

Consider two closely related yeasts *Saccharomyces cerevisiae* and *S. paradoxus*; both have two mating types. Molecular studies suggest sex is exceedingly rare, with estimates of an outcrossing event once per $5,000$ asexual divisions [61] or sex between once in every $1,000$ to $3,000$ generations [66]. For *C. reinhardtii* molecular studies suggest that just 1000 outcrossing events may have occurred in isolates sampled over the last 70 years [37]. Assuming $(1 - c) = 1/1000$ ($c = 0.999$), $10^7 \geq N_e \geq 10^6$ and $10^{-6} \geq m_g \geq 10^{-8}$ (a new mating type once every 1 to 100 million generations), the model predicts $13 \geq M \geq 3$ in the absence of viability selection treating the mating types unequally (see Fig. 4). Although this exceeds the two types observed in *Saccharomyces* and *Chlamydomonas*, our model shows that small mortality differences (of the order 0.1%) can reduce the number of mating types to 2 (see Section S5; also see below for other potential factors, e.g. species-specific genetic architecture).

Ciliates appear to reproduce sexually more often, though still infrequently; sex is limited by an immature period of $40 - 100$ asexual divisions [12, 59]. Studies on wild populations [12, 43] estimate sex to occur once in every few hundred generations. We predict that more frequent sex leads to more mating types: assuming $10^7 \geq N_e \geq 10^6$ and $10^{-6} \geq m_g \geq 10^{-8}$ but now setting $(1 - c) = 1/200$, the model predicts $28 \geq M \geq 6$ (see Supplementary Information). Known numbers of mating types are $5 - 13$ for *Euplotes*, 2 for *Aspidisca*, and $3 - 9$ for *Tetrahymena* [21, 59].

Most species of mushroom-forming fungi *Agaricomycotina* (a subdivision of the *Basidiomycota*) are obligately sexual [51]. *S. commune*, ($M > 23,000$) belongs to this family, and molecular evidence suggests it is one of the most sexual species in the fungal kingdom [53], with high mutation rates [2]. The fungal kingdom, in general, offers evidence of N_e covarying positively with numbers of mating types [36].

Focusing on *S. commune* and assuming obligate sex, a large effective population size ($N_e = 10^7$) and large mutation rate ($m_g = 10^{-6}$), we predict $520 \geq M \geq 420$ (see Fig. 4), well below the $23,000$ mating types known to exist. *S. commune* offers good biological reasons for our model underestimate; we assumed a single mating type locus. *S. commune* has tetrapolar mating type determination, with each type defined by two loci, each with two weakly recombining regions [38]. A mating type is not extinct when its genotype frequency reaches zero, but when one of its mating type alleles is lost. Extinctions become less likely in a system where the mating type allele is carried by many more individuals than the genotype.

Generally, multiple loci are expected to stabilize multi-mating type systems better than multiple alleles at a single locus ([9, 52, 69]). Multiple loci indeed frequently determine isogamous species mating types, with the gain or loss of loci causing mating type number transitions (e.g. *Paramecium bursaria* [59], *U. hordei*, *M. globosa*, *C. neoformans* [40]). Single-locus determination of more than 2 mating types is only common when mating types are determined at the diploid stage. The disassociation between mating type alleles and mating types then resembles that of multi-locus systems.

More drastic departures from our theory are possible due to genetic architecture. While the highly sexual *S. commune* has thousands of mating types, other highly sexual fungal species have abandoned mating types altogether. Homothallism in *Ascomycetes* [71] makes them lack bifactorial mating type determination, potentially limiting the scope for novel mating types [52]. Aligning homothallism with our model is possible if high rates of sex (observed in these taxa, suggesting an ecology that favours high recombination) would permit large numbers of mating types, but this route is mutation-limited. Mutations for self-compatibility then offer an alternative route to maintain frequent sex.

Turning to frequency-independent success differences between mating types, the model predicts that the signals

of such differences should manifest more strongly when sex is rare. Consequently, these signals are best sought during periods of asexual reproduction. In *Chlamydomonas*, bouts of asexuality frequently sweep single mating types to fixation [4], as a result of hitch-hiking on beneficial mutations and asexuality maintaining the linkage. Similar dynamics occur in fungi [46, 63], where amongst pathogenic species [8, 39, 49] there is also evidence for fitness differences between mating type alleles [14].

Our model could be criticized where it predicts two mating types, for lacking a mechanism (e.g. Fisherian sex ratio theory) that would prevent strong fluctuations around a 1 : 1 mating type ratio; here the parameter regime places the system precariously near the loss of all but one of the types. We consider these dynamical features real rather than a flaw. A common type is bound to have greatly diminished reproductive success under Fisherian dynamics with sex assumed obligate. Facultativeness of sex, however, means it can still reproduce (for other routes to deviations from 1 : 1 see [70] and [57]). Balancing selection is not always sufficient to maintain equal mating type ratios in *Coccidioides* [44] ($M = 2$) or *Dictyostelium discoideum* [13] ($M = 3$). In ciliates, at least two species of *Tetrahymena* have lost mating types [59] (*T. elliotti* and *T. pyriformis*). In the fungal kingdom, *Batrachochytrium dendrobatidis* only has a single mating type allele [40]. While contributions of drift and selection are hard to disentangle for each case, our model suggests that they may act synergistically to reduce the number of mating types.

II. DISCUSSION

Why do isogamous species have few (and species-specific numbers of) mating types, when the naïve prediction is that rare types should always invade? Our model contributes towards understanding this discrepancy through two important interacting ingredients: finiteness of populations (genetic drift) and facultative sex. These change the prediction from unbounded increases in the number of mating types (the prediction in infinite populations) to a species-specific number of mating types. This number is reduced if mutations yielding new mating types are rare, populations small, and sexual reproduction rare (compared with the number of asexual cycles).

Our model derives precise expectations for the effects of drift (discussed by [7, 13, 35]) across scenarios that differ in their rates of sex and effective population sizes, when all mating types are equally fit and when they are not. When equally fit, the model is a null model with no ecological differences beyond frequency dependent selection favouring rare types. Assessing drifts role in populations that can undergo asexual as well as sexual cycles [17, 62] necessitates a stochastic (rather than deterministic [35, 48]) modelling approach; we derive the null expectations without having to rely on simulations [17] while avoiding mathematical inconsistencies of earlier studies of plant SI systems [73].

Our generalized model includes frequency-independent fitness differences between mating types. Here, simulations show that small fitness differences are sufficient to further reduce the number of mating types when sex is rare. Note that the mechanisms incorporated in our model and others (non-mass-action mating kinetics [35], pheromone signalling [22, 30], organelle inheritance [32]) are not mutually exclusive. We expect drift to be important because isogamous sex is typically facultative, and this potentially enhances any mechanism evoked to constrain the success of rare types. The stronger fluctuations in mating type ratio caused by facultative (rather than obligate) sex also help understand the evolution of solutions to mate-finding difficulties: mating type switching or homothallism [25, 71]. Incorporating drift under facultative sex to models devoted to understanding the significance of such mechanisms appears fruitful: low rates of sex may e.g. decrease the costs of biparental inheritance (vegetative segregation generates homoplasmy) and amplify mate-finding problems [54].

Our model allows us to paint the following picture. Ecological conditions select for high or low rates of sex in a given facultatively sexual species [3, 5]. All else being equal, higher rates reduce drift, permitting more mating types to coexist at an evolutionary equilibrium. While available data fits our model qualitatively, other factors will play a role in the diversity of sexual strategies across taxa. Above we discussed homothallism; another route to a fundamentally different arrangement is male/female dimorphism (anisogamy), in which the number of sexes will be two based on different processes than the ones we envisage. Anisogamy models routinely produce only two size classes [65] (sperm and egg), and as it is advantageous to prevent sperm attempting fusing with other sperm (as neither gamete would provide sufficient cytoplasm for future development), it is logical to suspect that mating types become associated with size-based classifications of gametes (molecular evidence: [16]).

III. METHODS

For notational convenience, we initially set $M_{\max}$ as the maximum number of possible mating types. The vector $\boldsymbol{n}$, which describes the number of individuals of each mating type, is then of length $M_{\max}$. We denote by $T(\boldsymbol{n}'|\boldsymbol{n})$ the probability per unit time of transitioning from a state $\boldsymbol{n}$ to state $\boldsymbol{n}'$. In general, the probability $P(\boldsymbol{n}, t)$ of being in a state $\boldsymbol{n}$ at time t is given by

$$\frac{dP(\boldsymbol{n},t)}{dt} = \sum_{\boldsymbol{n}'} \left[T\left(\boldsymbol{n}|\boldsymbol{n}'\right) P\left(\boldsymbol{n}',t\right) - T\left(\boldsymbol{n}'|\boldsymbol{n}\right) P\left(\boldsymbol{n},t\right)\right] . \tag{5}$$

In order to align this mathematical formulation with the model described in the main text, we must define the

terms $T(\boldsymbol{n}|\boldsymbol{n}')$, which in the Moran model consists of the birth of one individual and death of another (see Eqs. (1-2));

$$\begin{aligned}\mathcal{T}_{ij} &\equiv T\left[(\ldots, n_i+1, \ldots, n_j-1, \ldots)\,|\,(\ldots, n_i, \ldots, n_j, \ldots)\right] \\ &= c\frac{n_i}{N}D_j\frac{n_j}{N} + \left(\frac{1-c}{2}\right)\frac{n_i}{N}\left(\frac{N-n_i}{N}\right)D_j\frac{n_j}{N} \\ &\qquad\qquad \text{if} \quad n_i > 0\,, \quad (6) \\ &= \left(\frac{m}{M_{\max}-M}\right)\frac{n_j}{N} \qquad \text{if} \quad n_i = 0\,. \quad (7)\end{aligned}$$

Here M is the number of mating types present in the population (i.e. the number of non-zero entries in $\boldsymbol{n}$) and the term $1/(M_{\max}-M)$ is a normalization factor that accounts for the fact that a new mutation may be assigned to any of the unoccupied mating type labels. Note that we have used the property that $\sum_{i=1}^{M_{\max}} n_i = N$ to simplify Eq. (1) to Eq. (6). Also note that in the limit $c \to 1$ the model simplifies to the neutral infinite allele Moran model with mutation. A similar modelling approach has been used to investigate the number of SI alleles in plants [17, 72, 74], but focused on diploid systems and without accounting for asexual reproduction. Meanwhile our generic mating kinetics (mass action) are the same as those used in the first of four models explored in [35].

The time for N reproduction events is approximately N in units of t (see Section S2). We therefore introduce τ as the generation time, $\tau = t/N$. In a similar fashion we introduce $m_g = mN$ as the per-generation mutation rate. With $c = 1$ this model reduces to the infinite allele neutral Moran model [11]. Our model does not account for the possibility that a gamete chosen for sexual reproduction fails to find a mate, unlike models described in [35] (see "Mating Kinetics" 2 and 3). Finally note that Eq. (6) implicitly assumes that sexual reproduction events are not temporally correlated. For an alternative approach see [25].

To obtain the deterministic (infinite population size) limit to the dynamics, we can apply a diffusion approximation [11]: assuming large N, we transform into the approximately continuous variables $x_i = n_i/N$, Taylor expand Eqn. (5) in N^{-1} and take the limit $N \to \infty$ [45]. Recall that the values D_i are chosen from a normal distribution with mean 1 and standard deviation σ. We can therefore rewrite these terms as $D_i = 1 + \sigma d_i$, where d_i is normally distributed with mean 0 and standard deviation 1. Assuming σ is small and that the mutation rate is much smaller, we obtain the description (see Section S2)

$$\begin{aligned}\frac{\mathrm{d}x_i}{\mathrm{d}\tau} &= \frac{(1-c)}{2}\left[\sum_{j=1}^{M_{\max}} x_i x_j (x_j - x_i)\right] \\ &\quad +\sigma\sum_{j=1}^{M_{\max}} x_i x_j \left[\left(\frac{1-c}{2}\right)(d_i x_j - d_j x_i)\right. \\ &\quad \left. +\left(\frac{1+c}{2}\right)(d_j - d_i)\right]. \qquad (8)\end{aligned}$$

These dynamics, illustrated in Fig. 3, recapitulate those of "Mating Kinetics 1" in [35] when $c = 0$ and $\sigma = 0$.

When $\sigma = 0$, given an initial number M_0 mating types present in the population, a fixed point exists at $x_i = 1/M_0$ for the present types and $x_i = 0$ otherwise. This situation represents even mating type ratios. Considering just the present mating types, the fixed point has eigenvalues

$$\lambda_i = -\frac{1-c}{2M_0}\,, \qquad (9)$$

and is therefore stable as long as there is at least some sexual reproduction, i.e. $c < 1$ (recall that if $c = 1$ the model become the neutral infinite allele Moran model, in which the dynamics are governed entirely by drift). If a novel mating type is introduced into the population, this fixed point becomes unstable, and a new fixed point arises at $x_i = 1/(M_0+1)$. The stability of the fixed point decreases with increasing c and M_0. However, since it is always stable for $c < 1$, the number of mating types is expected to grow linearly with time;

$$M(\tau) = M_0 + m_g\tau\,. \qquad (10)$$

If $\sigma > 0$, a fixed point at even sex ratios is no longer possible. The fixed point is dependent on the stochastically-chosen values of $\boldsymbol{d}$, and is given by

$$x_i = \frac{1}{M_0} + \sigma\frac{1-c-M_0-cM_0}{(1-c)M_0^2}(M_0 d_i - \sum_{j=1}^{M_{\max}} d_j) \qquad (11)$$

for each type present in the population. Note that when c, M_0 and σ are small (frequent sex, a low number of mating types and small variance in mating type fitness) the deviation from even sex ratios is small. However, as c, M_0 and σ increase, deviations from even sex ratios become more pronounced. This additionally leads to an increased probability that the interior fixed point containing all M_0 mating types becomes unstable (or disappears entirely) however we do not analytically quantify this effect here.

We next aim to analytically characterize the probabilistic dynamics. For this we wish to obtain the stationary probability distribution $P^{\mathrm{st}}(\boldsymbol{n})$, which is the solution to Eq. (5) in the limit $t \to \infty$. In the Supplementary Information, we show that this equation can be solved, but only if there are no frequency-independent fitness differences between the mating type alleles ($\boldsymbol{D} = \boldsymbol{1}$ or

equivalently $\sigma = 0$). Under these conditions the probability transition rates, Eqs (6-7) can be decomposed into products of birth and death rates that depend only on the number of the individuals belonging to the mating types that increase and decrease;

$$\mathcal{T}_{ij} = b(n_i)d(n_j) \tag{12}$$

where

$$b(k) = \overbrace{c\left(\frac{k}{N}\right)}^{\substack{\text{Asexual}\\ \text{reproduction}}} + \overbrace{\left(\frac{1-c}{2}\right)\left(\frac{k}{N}\right)\left(\frac{N-k}{N}\right)}^{\substack{\text{Sexual}\\ \text{reproduction}}} \quad \text{if} \quad k \geq 1\,,$$
$$b(0) = \underbrace{\left(\frac{m}{M_{\max}-M}\right)}_{\text{Mutation}} \qquad d(k) = \underbrace{\left(\frac{k}{N}\right)}_{\text{Death}} \quad \forall \quad k\,. \tag{13}$$

Given the functional form of Eq. (12), the stationary distribution can be shown to be given by (see Supplementary Information);

$$P^{\text{st}}(\boldsymbol{n}) \propto \prod_{i=1}^{M-1}\prod_{k=0}^{n_i^{\downarrow}-1} \frac{b(k)d\left(N-k-\sum_{j=1}^{i-1}n_j^{\downarrow}\right)}{b\left(N-k-\sum_{j=1}^{i-1}n_j^{\downarrow}-1\right)d(k+1)}\,, \tag{14}$$

where $\boldsymbol{n}^{\downarrow}$ is the vector $\boldsymbol{n}$ with its elements rearranged in descending order and M is the number of non-zero elements of $\boldsymbol{n}$ (see section S1). To our knowledge this is the first time this result has appeared in the literature. In a distinct model set-up, a similar result has been obtained [47]; as this result also relies on transitions having forms of the type given in Eq. (12), the expressions may be relateable. Eq. (14) perfectly captures the results of simulations (see Figs. S2-S4). Classic investigations into the number of SI alleles in plants also rely on calculating the stationary distribution of alleles. However they do so having applied a diffusion approximation and consider the interactions between a single focal SI allele and a population fixed at some prescribed frequency distribution, an approach criticized by Moran based on its fundamental mathematical inconsistencies (see [73] for discussion). This approach was necessary as the models of SI alleles in plants feature diploid sex determination, and so transitions do not follow the functional form given in Eq. (12).

We are interested in the stationary distribution of the number of mating types present in the population $\mathcal{P}^{\text{st}}(M)$, which is related to $P^{\text{st}}(\boldsymbol{n})$ by

$$\mathcal{P}^{\text{st}}(M) = \sum_{\boldsymbol{n}\in S^{(M)}} P^{\text{st}}(\boldsymbol{n})\,. \tag{15}$$

where $S^{(M)}$ is the set of all vectors $\boldsymbol{n}$ that contain M non-zero elements. Since this expression is unintuitive, we proceed to characterize the mode of $\mathcal{P}^{\text{st}}(M)$. The calculation is described in full in Section S4. To begin we note that if N is large, and $m_g = mN$ small, then $P^{\text{st}}(\boldsymbol{n})$ will consist of a series of peaks, each located at states where the frequency of the mating types is approximately given by the deterministic fixed points. The mode of $P^{\text{st}}(\boldsymbol{n})$ can then be obtained by considering its values at states $\boldsymbol{n}$ in the proximity of successive fixed points; $n_i \approx N/M$ for M mating types and zero otherwise. Obtaining the mode of $\mathcal{P}^{\text{st}}(M)$ is more complicated; it depends on values of $P^{\text{st}}(\boldsymbol{n})$ far from the fixed points (see Eqn. (15)). However we can consider limiting behaviour of $P^{\text{st}}(\boldsymbol{n})$ to calculate upper and lower bounds of the mode of $\mathcal{P}^{\text{st}}(M)$, which simulations tell us typically lies close to the mean (see Supplementary Information). For a lower bound, we assume that $P^{\text{st}}(\boldsymbol{n})$ is constructed from a series of delta peaks at the deterministic fixed points. For an upper bound, we assume that $P^{\text{st}}(\boldsymbol{n})$ is completely flat in the region around the deterministic fixed points. The full calculation is detailed in the Section S4. For a general facultatively sexual system, the upper and lower bounds of the mode of $\mathcal{P}^{\text{st}}(M)$ can be be shown to be given by (see Section S4.2.3)

$$M^*_{\text{UB}} > \text{Mode}\left[\mathcal{P}^{\text{st}}(M)\right] > M^*_{\text{LB}}\,, \tag{16}$$

where M^*_{UB} and M^*_{LB} can be obtained as solutions to the equations

$$R(M^*_{\text{LB}}) = 1\,, \quad \text{and} \quad \left(\frac{N}{M^*_{\text{UB}}-1}-1\right)R(M^*_{\text{UB}}) = 1\,, \tag{17}$$

with

$$\begin{aligned} R(M) = 2m(1+c)^{-\frac{1}{2}+\frac{1+c}{1-c}N} M^{-1+\frac{3}{2}M+\frac{1+c}{1-c}MN-N} \times \\ (M-1)^{-\frac{3}{2}(M-1)+\frac{2-cM-M}{1-c}N} \times \\ (cM+M-2)^{\frac{1}{2}(M-1)-\frac{2-cM-M}{1-c}N} \times \\ (cM+c+M-1)^{-\frac{M}{2}-\frac{1+c}{1-c}MN+N}\,. \end{aligned} \tag{18}$$

A numerical solution to this equation for a given set of parameters can be obtained quickly using a standard root-finding algorithm. Comparing analytical results with those from stochastic Gillespie simulation [18] with $\sigma = 0$, we find excellent agreement (see Figs. S6-S8).

Code availability statement

The stochastic simulations conducted in this paper is available at:
https://github.com/gwaconstable/FiniteNMatingTypes

Data availability statement

Data generated during the study is available at:
https://github.com/gwaconstable/FiniteNMatingTypes

Contributions

G.W.A.C. designed the project and conducted the mathematical analysis. G.W.A.C. and H.K. developed the model and wrote the paper.

Competing interest

The authors declare no competing interests.

Funding

G.W.A.C. thanks the Finnish Center for Excellence in Biological Interactions and the Leverhulme Early Career Fellowship provided by the Leverhulme Trust for funding. H.K. thanks the Swiss National Science Foundation and the Academy of Finland for funding.

Acknowledgements

We thank Joshua Christie, Leslie Turner and the audience of the seminar series at the Milner Centre for Evolution for useful discussions and input.

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